Remarks/Arguments

Claims 39-54, previously presented, with claims 43, 48, and49 amended hereby, are pending.

According to the Office Action, claims 43, 44, and 48 are apparently considered indefinite; however, the Office Action contains no rejection under 35 USC 112, second paragraph. Nonetheless, claims 43, 44, and 48 are amended, hereby, in order to expedite prosecution by addressing the issues raised in the Office Action as concerns these claims being allegedly indefinite.

Claims 39-54 stand rejected under 35 USC 101 and under 35 USC 112, first paragraph, for allegedly lacking credible utility. Reconsideration of these rejections is requested.

According to the statement of rejection (Office Action page 3):

> The instant specification, as filed, discloses that Seladin-1 is expressed throughout the human brain and other human tissues of normal and AD [Alzheimer's Disease] patients and that the transcripts levels of Seladin-1 "were significantly lower in brain regions with severe neurodegeneration" as well as that "in AD brains, the expression of Seladin-1 was substantially lower in the inferior temporal lobe compared to the frontal cortex" (page 25-26, Figures 17 and 18). Thus, there is no disclosure that the claimed polynucleotides are expressed at altered levels or forms [*sic* form] in any specific, diseased tissue or otherwise associated with Alzheimer's disease, as implied by the instant specification. Therefore, one would reasonably conclude that based on the fact pattern of tissue distribution[,] Seladin-1 cannot be used as a marker for Alzheimer's Disease and, further, one would not logically accept that [the] biological function of Seladin-1 is related to its protective role against degeneration and cell death in general, as asserted in the instant specification.

Notwithstanding the foregoing statements to the contrary, the subject application, as originally filed, satisfies the requirements for utility and enablement of use under §101 and §112, first paragraph.

First of all, applicants incorporate herein by reference their arguments contained in the remarks to the Supplemental Amendment filed January 25, 2004. Additionally, the following

remarks are provided, which further demonstrate that the subject application, as originally filed, satisfies the requirements for utility and enablement of use.

Secondly, the statement of rejection sets forth a "hypothetical example" of adisclosure that satisfies the utility requirements under §101 and §112, ¶1, i.e.:

> According to legal standards, a specification can meet the requirements of utility and enablement for a new polypeptide or polynucleotide as long as the specification discloses at least one credible, specific and substantial asserted utility for the new DNA or protein encoded thereby, or a well-established utility for the claimed sequences would be *prima facie* obvious to the skilled artisan (*emphasis in original*). A hypothetical example may serve to clarify. For example, a hypothetical specification discloses that a claimed polynucleotide is expressed in colon cancer and not expressed in healthy colon tissue. The hypothetical specification does not disclose the biological activity of the polypeptide encoded by the polynucleotide. The claimed polynucleotide in the hypothetical example would not be rejected under 35 U.S.C. §§101 and 112, first paragraph, as it has utility and is enabled as a colon cancer marker.

Figures 4, 5, 16, and 17 of the subject application clearly show transcript levels of Seladin-1 are significantly lower in brain regions evidencing neurodegeneration, i.e., in the temporal lobe of AD patients, but not in healthy control persons. The temporal lobe is much more affected by AD (i.e., much more cell death of neurons occurs) than the frontal lobe. The level of Seladin-1 correlates with this phenomenon. The same level of Seladin-1 in the frontal lobe of AD patients compared to control persons reflects that the frontal lobe of AD patients shows only minor signs of neurodegeneration in AD patients. Thus, where profound neurodegeneration (i.e., cell death) occurs, the Seladin-1 level is low; however, where there is nearly no cell death, the level of Seladin-1 is high. Thus, the claimed polynucleotides are expressed at altered levels in diseased tissue of AD patients compared to the normal tissue of control persons.

Contrary to the allegations found in the statement of rejection, the aforesaid information is disclosed in the specification and, thus, credible utility is asserted. Further, the altered expression levels clearly reflect the cell-protective function and biological role of Seladin-1.

Further disclosure of the function of Seladin-1 and its biological activity is provided by application Figure 20. Figure 20 also shows that Seladin-1 functions as an oxido-reductase, conferring resistance against induction of cell death, which underscores the protective function of Seladin-1.

Application Figures 12 to 15 describe Seladin-1 in detail and Figure 18 and Example 1 evidence the cloning of Seladin-1; i.e., Seladin-1 is cloned and sequenced and recombinantly expressed in tissue culture cells.

Thus, the subject application discloses the credible utility necessary to satisfy the utility standard under §101 and §112, first paragraph, as set forth in the statement of rejection.

Claims 40-45, and 53 stand rejected under 35 USC 112, first paragraph, as allegedly lacking descriptive support in the subject application as originally filed (a new-matter rejection). Reconsideration is requested.

Note is taken of the various court decisions cited in the statement of rejection. None of the cited decisions is on point with the instant facts.

As opposed to the facts on which the findings in the cited court decisions were made, the subject application discloses both the *structure* of the claimed *protein molecule* and the *structure* of

the claimed *nucleic acid* encoding the protein molecule. Accordingly, none of the decisions cited in the statement of rejection is controlling under the present facts.

The issue raised in the new-matter rejection concerns the claimed "functional variant" of the *structurally disclosed* protein molecule, i.e., the 516 amino-acid-containing-protein structure disclosed in SEQ ID NO: 1 and the nucleic acid molecule encoding the protein as disclosed in SEQ ID NO: 2. The person of ordinary skill in the art would recognize that applicants had possession of the presently claimed subject matter, including a functional variant of the disclosed protein molecule.

Favorable action is requested.

Respectfully submitted,

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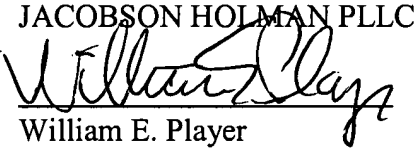
By 
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